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Relationship of venous blood gas with cervical esophagogastric anastomotic leak

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Abstract

Objective: this study investigated the relationship between various parameters of venous blood gas analysis of gastric fundus veins and cervical esophagogastric anastomotic leaks after transhiatal esophagectomy.

Background: decreased tissue perfusion is one of the causes of anastomotic leak. There are various methods used to assess gastric conduit perfusion, with different results, and we lack a reliable method.

Method: this descriptive study, performed from March 2008 to October 2010, consisted of 45 patients with esophageal cancer who underwent transhiatal esophagectomy. After gastrolysis, blood samples were taken from a gastric fundus vein and submitted for venous blood gas analysis. The cervical wounds were examined 5 days postoperatively. The patients were divided into 2 groups based on the presence of leakage, and mean values of the venous blood gas analysis were compared.

Results: we observed significant differences in mean pH, PCO_2, and O_2 saturation between the 2 groups ($p = 0.04$, $p = 0.03$, and $p = 0.04$, respectively).

Conclusion: venous blood gas analysis of gastric fundus veins appears to be a feasible and fast method for intraoperative assessment of microperfusion in the gastric fundus.

Keywords

Anastomotic leak, blood gas analysis, esophageal neoplasms, esophagectomy, postoperative complications

Introduction

After esophageal resection, reconstruction of the upper gastrointestinal tract may be achieved by gastric transposition and cervical esophagogastrostomy.[1,2] Pulling the stomach up to the neck as an esophageal substitute is successful because of its extraordinary plasticity and stretchability as well as the richness of its submucosal vascular network.[3–6] With the rediscovery of transhiatal esophagectomy (THE) without thoracotomy, cervical esophagogastric anastomosis (CEGA) has become an increasingly common procedure.[7] One advantage of this approach is that a CEGA leak is seldom associated with mediastinitis. In addition, more than 98% of CEGA leaks are relatively benign and can be managed successfully with local wound care. However, there is a small incidence of disastrous complications associated with CEGA leakage.[8] As many as 50% of CEGA leaks result in anastomotic stricture as fibrosis associated with healing becomes established.[9] Only a few reports have attempted to identify risk factors for the CEGA leak. Intuitive preoperative risk factors (preexisting diabetes, cardiovascular disease, and smoking history) potentially reduce microperfusion of tissues.[10,11] Because the short gastric and left gastric arteries are divided in the conduit mobilization, the tip of the conduit is supplied by collateral vessels from the right gastric and right gastroepiploic arteries, which may lead to

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decreased gastric conduit perfusion.[12] Decreased gastric conduit perfusion and consequent relative ischemia of the tip of the newly mobilized gastric conduit in CEGA has long been implicated in a higher rate of leakage compared to an intrathoracic anastomosis. Different results have been obtained with various methods of assessing gastric conduit perfusion: optical fiber spectroscopy, visible light spectroscopy, the combination of a laser Doppler flowmeter and spectrophotometer, a laser Doppler imager, partial tissue oxygen pressure with a Clark-type polar graphic oxygen electrode, continuous measurement of mucosal PCO_2 using recirculation gas analysis with a TONOCAP device together with mean arterial pressure measurement, and cardiac output and systemic vascular resistance by pulse contour analysis.[13–16] Thus we lack a reliable method to assess gastric conduit perfusion. In this study, we assessed the relationship between various parameters of venous blood gas analysis of gastric fundus veins after gastrolysis and CEGA leaks in the neck after THE.

Patients and methods

The participants of this descriptive prospective study, performed at Al-Zahra Hospital, comprised all patients with esophageal cancer who were considered for THE surgery between March 2008 and October 2010. The study was confirmed by the ethical committee of Isfahan University of Medical Sciences, and all patients gave informed consent. The procedure was THE without thoracotomy and a CEGA with the stomach placed in the posterior mediastinum. There were 47 patients who met the criterion. Patient data including sex, age, type of the tumor (squamous cell carcinoma or adenocarcinoma), hypertension, respiratory insufficiency, cardiovascular disease, history of gastric surgery, history of neoadjuvant therapy, and serum albumin before surgery (above and below $3\,g{\cdot}dL^{-1}$) were collected.

All patients were operated on by the same surgeon. During the operation, after gastrolysis and before anastomosis of the esophagus with the gastric conduit, blood samples were taken simultaneously from veins of the gastric fundus and peripheral veins, using a thin needle, and sent for venous blood gas analysis by a blood gas analyzer model AVL993. The data of pH, PCO_2, O_2sat, PO_2, base excess, and HCO_3^- were gathered. Cervical wounds were examined for wound leakage every day for 5 days postoperatively. Patients without leakage were instructed to drink 250 mL of methylene blue solution to check for an exit of dye from the wound. The patients were divided into 2 groups: group A (leakage) and group B (no leakage). The management in all cases of leakage included reopening the wound and frequently changing the dressing, which has been reported to yield good results.[17] Samples of the border of the esophageal were evaluated by a pathologist for microinvasion of tumor cells, and the results were recorded. The patients were followed up for 4 weeks postoperatively.

Data are given as mean ± standard deviation. Patient data and the blood gas parameters were analyzed with SPSS software (SPSS, Inc., Chicago, IL, USA), using Student's *t* test and Fisher's exact test.

Results

Two (4.25%) patients died before the 5th postoperative day, leaving 45 included in the analysis. The mean age of these 45 patients was 61 ± 12 years and 32 (71.1%) were male. Forty (89%) patients had squamous cell carcinoma and 5 (11%) had adenocarcinoma. No patient had history of gastric surgery, and none had malignant cell involvement in the margin of the esophageal sample. Of the 45 patients, 12 (group A, 26.7%) had leakage of the anastomosis and 33 (group B, 73.3%) had no leakage. In group A, all patients had clinically silent leaks. Based on Fisher's exact test, there were no significant differences between the 2 groups with respect to mean PO_2 ($p = 0.86$), base excess ($p = 0.8$), and HCO_3^- ($p = 0.55$) in peripheral venous blood, as well as hypertension ($p = 0.99$), diabetes mellitus ($p = 0.99$), respiratory insufficiency ($p = 0.55$), cardiac diseases ($p = 0.99$), neoadjuvant treatment ($p = 0.99$) and serum albumin $<3\,g{\cdot}dL^{-1}$ ($p = 0.99$). On the other hand, mean pH, PCO_2, and O_2sat of venous blood from the gastric fundus were significantly different between the 2 groups, according to Student's *t* test (Table 1). At 4 weeks postoperatively, no patient with leakage had developed dysphagia.

Table 1. Venous blood gas parameters of gastric fundus in patients with and without anastomotic leakage.

Variable	Leakage (Group A)	No leakage (Group B)	*p* value
No. of patients	12	33	
Mean pH	7.2 ± 0.08	7.39 ± 0.09	0.04
Mean PCO_2	46.0 ± 8.6	41.1 ± 9.7	0.03
Mean O_2sat	55.0 ± 20	66.6 ± 17.2	0.04
Mean PO_2	45.0 ± 15.2	47.9 ± 24.6	0.7
Mean base excess	−8 ± 2.9	−6.8 ± 3.2	0.24
Mean HCO_3^-	18.36 ± 2.3	18.37 ± 2.5	0.99

Discussion

Based on our study findings, the THE operative mortality rate was 4.25%, nearly the same as that of similar hospitals with surgeons experienced in THE. However, the rate of CEGA leak in this study was higher than those obtained in other studies, although there was no mortality associated with cervical leakage. A CEGA leak is among the leading causes of preoperative morbidity and mortality after esophagectomy. Operative mortality has been shown to be inversely related to the surgeon's experience and hospital volume. In a retrospective review of esophagectomies performed for cancer, 42 patients were operated on by surgeons who performed 6 or more esophagectomies per year, and 32 were operated on by surgeons who performed 5 or fewer esophagectomies per year; surgeons experienced in performing this operation had a significantly lower operative mortality (0% vs. 7%, $p < 0.001$). In a retrospective study determining the correlation between hospital volume and operative mortality of esophagectomy, operative mortality was 17.3% in low-volume hospitals compared to 3.4% in high-volume hospitals.[18]

In our study, the anastomotic leak rate for cervical anastomosis was 26.7%. There is no universally accepted definition of anastomotic leak. The definitions and values used to measure anastomotic failure vary extensively and preclude accurate comparison of rates obtained by different studies and institutions. In a meta-analysis of the literature on surgical treatment of patients with esophageal carcinoma by Muller and colleagues,[19] the anastomotic leak rate for cervical anastomosis was $11\% \pm 6\%$, and mortality associated with a cervical leak was $20\% \pm 11\%$. The higher CGEA leak rate in our patients may be due to accurate detection of leaks that may be unimportant and usually heal spontaneously, so all the patients managed with reopening the wound and frequently changing the dressing had good results and no mortality. Our study could not demonstrate that neoadjuvant treatment, serum albumin $<3\,g{\cdot}dL^{-1}$, diabetes mellitus, heart disease, chronic renal insufficiency, hypertension, or chronic obstructive pulmonary disease could be considered independent risk factors for anastomotic leak. This may be due to the small number of cases.

As perfusion of the tissues is reduced, cells are deprived of oxygen and must switch from aerobic to anaerobic metabolism. The product of anaerobic respiration is not carbon dioxide but lactic acid. When enough tissue is underperfused, accumulation of lactic acid in the blood produces systemic metabolic acidosis. In our study, the mean pH of blood of gastric fundus veins in the anastomotic leakage group was significantly lower than that of the group without leakage. Clearly, decreased tissue perfusion causes cell metabolism to change from aerobic to anaerobic, with acidic products of metabolism. So it seems reasonable that declining pH in the venous blood of the gastric fundus could be a reflection of reduced gastric perfusion, a known risk factor for anastomosis leakage. Based on this study, mean O_2sat in the leakage group was significantly lower; therefore, lower O_2sat in gastric fundus venous blood might reflect tissue ischemia, and could be a predictor of anastomosis leakage. On the other hand, we found that the mean PCO_2 in gastric fundus venous blood in the leakage group was significantly higher. Impaired venous drainage is one of the possible causes of anastomotic leakage, which is also emphasized in this study. Differences between mean PO_2, base excess, and HCO_3^- were not significantly different between the 2 groups in this study.

The results showed that although there were no significant difference in some factors in gastric fundus venous blood after gastrolysis between the 2 groups, in addition to the technique employed and experience of the surgeon, some factors could predict the likelihood of anastomotic leakage. Impaired venous drainage seems to be one of the important factors for anastomosis leakage. If we are able to predict the possibility of ischemic tissue and impaired venous drainage, the surgeon can change the surgical technique and/or infuse nitroglycerin to reduce the risk of anastomosis leakage. Venous blood gas analysis of a gastric fundus vein might be considered a feasible and fast method for intraoperative assessment of microperfusion of the gastric fundus in esophageal anastomosis. Further clinical studies could help us define its role in the prediction of anastomotic leakage.

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Conflicts of interest statement

None declared.

References

1. Casson AG, Porter GA and Veugelers PJ. Evaluation and critical appraisal of anastomotic technique following resection of esophageal adenocarcinoma. *J Thorac Cardiovasc Surg. Dis Esophagus* 2002; 15: 296–302.
2. Ellis Jr FH, Heatley GJ, Krasna MJ, Williamson WA and Balogh K. Esophagogastrectomy for carcinoma of the esophagus and cardia: a comparison of findings and results after standard resection in three consecutive eight-year intervals with improved staging criteria. *J Thorac Cardiovasc Surg* 1997; 113: 836–848.

3. Ercan S, Rice TW, Murthy SC, Rybicki LA and Blackstone EH. Does esophagogastric anastomotic technique influence the outcome of patients with esophageal cancer? *J Thorac Cardiovasc Surg* 2005; 129: 623–631.
4. Akiyama H, Miyazono H, Tsurumaru M, Hashimoto C and Kawamura T. Use of the stomach as an esophageal substitute. *Ann Surg* 1978; 188: 606–610.
5. Liebermann-Meffert DM, Meier R and Siewert JR. Vascular anatomy of the gastric tube used for esophageal reconstruction. *Ann Thorac Surg* 1992; 54: 1110–1115.
6. Koskas F and Gayet B. Anatomical study of retrosternal gastric esophagoplasties. *Anat Clin* 1985; 7: 237–256.
7. Orringer MB and Sloan H. Esophagectomy without thoracotomy. *J Thorac Cardiovasc Surg* 1978; 76: 643–654.
8. Iannettoni MD, Whyte RI and Orringer MB. Catastrophic complications of the cervical esophagogastric anastomosis. *J Thorac Cardiovasc Surg* 1995; 110: 1493–1501.
9. Orringer MB, Marshall B and Iannettoni MD. Eliminating the cervical esophagogastric anastomotic leak with a side-to-side stapled anastomosis. *J Thorac Cardiovasc Surg* 2000; 119: 277–288.
10. Cooke DT, Lin GC, Lau CL, Zhang L, Si MS, Lee J, et al. Analysis of cervical esophagogastric anastomotic leaks after transhiatal esophagectomy: risk factors, presentation, and detection. *Ann Thorac Surg* 2009; 88: 177–185.
11. Tabatabai A, Hashemi M, Mohajeri G, Ahmadinejad M, Khan IA and Haghdani S. Incidence and risk factors predisposing anastomotic leak after transhiatal esophagectomy. *Ann Thorac Med* 2009; 4: 197–200.
12. Pham TH, Perry KA, Enestvedt CK, Gareau D, Dolan JP, Sheppard BC, et al. Decreased conduit perfusion measured by spectroscopy is associated with anastomotic complications. *Ann Thorac Surg* 2011; 91: 380–385.
13. Karliczek A, Benaron DA, Baas PC, Zeebregts CJ, van der Stoel A, Wiggers T, et al. Intraoperative assessment of microperfusion with visible light spectroscopy in esophageal and colorectal anastomoses. *Eur Surg Res* 2008; 41: 303–311.
14. Bludau M, Vallböhmer D, Gutschow C, Hölscher AH and Schröder W. Quantitative measurement of gastric mucosal microcirculation using a combined laser Doppler flowmeter and spectrophotometer. *Dis Esophagus* 2008; 21: 668–672.
15. Schröder W, Beckurts KT, Stähler D, Stützer H, Fischer JH and Hölscher AH. Microcirculatory changes associated with gastric tube formation in the pig. *Eur Surg Res* 2002; 34: 411–417.
16. Schröder W, Stippel D, Gutschow C, Leers J and Hölscher AH. Postoperative recovery of microcirculation after gastric tube formation. *Langenbecks Arch Surg* 2004; 389: 267–271.
17. Patterson GA, Cooper JD, Deslauries J, Lerut AE (eds) *Pearson's Thoracic and Esophageal Surgery*. Volume 2, 3rd ed. Philadelphia: Churchill Livingstone, 2008, pp. 546–547.
18. Begg CB, Cramer LD, Hoskins WJ and Brennan MF. Impact of hospital volume on operative mortality for major cancer surgery [see comments]. *JAMA* 1998; 280: 1747–1751.
19. Müller JM, Erasmi H, Stelzner M, Zieren U and Pichlmaier H. Surgical therapy of oesophageal carcinoma [Review]. *Br J Surg* 1990; 77: 845–857.